Augmentation of ruptured tendon using fresh frozen Achilles tendon allograft in two dogs: a case report

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ABSTRACT: This article describes two cases of augmentation of ruptured tendon with fresh frozen Achilles tendon allograft (FFATA) in dogs. Case 1 was a two-year-old crossbreed dog (29 kg) that presented with an open wound on the right forelimb and with complete rupture of the *flexor carpi ulnaris* and superficial digital flexor tendons. Case 2 was a four-year-old crossbreed dog (4 kg) with partial ruptures of the patellar tendon and detachment of the tibial tuberosity in the right hind limb. In both cases, the ends of the ruptured tendon were sutured and apposed after debridement. To minimize suture failure, FFATA (cut to sufficient size) was placed across the primary suture with tension and sutured to the host tendon. In addition, Case 2 received a Krackow suture through a transverse bone tunnel made in the tibia to fix the patellar tendon along with the tibial tuberosity *in situ*. The surgical areas healed without any evidence of exaggerated inflammatory response or clinical signs consistent with rejection of the allograft. Both the dogs had normal ambulation and weight bearing on the affected limb 12 weeks postoperatively. No postoperative complications were observed during a one-year follow up period except for slight contracture of the carpus and digits of the affected limb in Case 1. Thus, ruptured tendons can be successfully repaired using suture and augmentation with FFCTA. Augmentation with FFATA may provide additional stability, which counters tension on the primary repair and reduces the chance of gap formation or suture failure in case of reconstruction of the damaged tendon in dogs.

Keywords: tendon rupture; Achilles tendon allograft; augmentation; dog

The tendon is a kind of musculoskeletal tissue that transmits force from muscle to bone. Lacerations, ruptures or inflammation of the tendon cause marked morbidity and can have a major impact on normal ambulation. The tendon consists of soft and fibrous connective tissue composed of densely packed collagen fibre bundles aligned parallel to the longitudinal tendon axis and surrounded by a tendon sheath. The well-organised tendon architecture supports large tensile forces and glides readily under tension to transmit force generated by muscle contraction to the bone to cause locomotion (James et al. 2008). Tendon ruptures in humans may occur without any obvious predisposing factors; however, local use of corticosteroids (Clark et al. 1995), and previous orthropaedic procedures are considered

to be risk factors (Jarvela et al. 2005; Shipov et al. 2008). Tendon rupture in dogs is generally the result of a direct trauma (Shipov et al. 2008).

The restoration of normal tendon function after injury requires reestablishment of tendon fibres and the gliding mechanism between the tendon and its surrounding structures (James et al. 2008). In most cases of tendon laceration or rupture, surgical intervention is required to direct the natural process of healing and occasionally the damage exceeds the natural regenerative ability even with existing treatment modalities. Tendon repair is a slow process that is complicated by the need to provide appropriate and timely tension to the repair tissue (James et al. 2008). Owing to the specialised nature of tissues, tendon regeneration has proved an elu-

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sive goal for tissue engineering. There is a significant clinical need for augmentative and substitutional approaches that enhance the structural performance of damaged and degenerated tendon tissues (Gemmill and Carmichael 2003; Kewa et al. 2011).

The most commonly used surgical technique for repair of ruptured tendon includes debridement and apposition of the severed ends with primary sutures. Suture patterns developed for this purpose include the Krakow suture, locking loop, Bun nell-Mayer and three-loop pulley patterns (Montgomery and Fitch 2003; Moores et al. 2004). However, in cases where excessive tension results from retraction of the muscle-tendon unit or extensive tendon damage leading to a significant decrease in tensile strength, tendon repair with suture alone may not be sufficient. Thus, augmentation of the injured tendon may be beneficial to provide addition stability, neutralise tension, decrease the risk of gap formation and minimise suture failure.

Tendon allografts play an important role in tendon and ligament reconstruction, particularly where there is a shortage of suitable available local tissue. The advantages of using allograft tissue include a lack of donor site morbidity, high tensile strength, decreased surgical time, smaller surgical incisions and a low risk of arthrofibrosis (Nellas et al. 1996; Robertson et al. 2006). The Achilles tendon (tendo calcaneus communis) is the strongest tendon in the structure of the musculoskeletal system in the dog. The Achilles tendon allograft (ATA) has been used mainly in human medicine for tendon and ligament reconstruction because of its good mechanical strength, and long and wide aponeurosis (Gasser and Uppal 2006; Kuhn and Ross 2007). However, until now literature pertaining to the augmentation and repair of ruptured tendons using a fresh frozen Achilles tendon allograft (FFATA) in clinical cases in veterinary practice is scanty. The purpose of this case report is to describe the clinical management and surgical outcomes of augmentation of ruptured flexor carpi ulnaris and superficial digital flexor tendons in one dog, and a partial tear of the patellar tendon with extensive tissue damage along with detachment of tibial tuberosity in another dog using FFATA.

Case description

Case 1 was a two-year-old intact male crossbreed dog weighing 29 kg that presented with a lacerated wound on the right forelimb. Physical examination revealed an open laceration on the caudomedial surface of the right forelimb 3 cm proximal to the carpal pad. The wound area was shaved and scrubbed using chlorhexidine solution and the wound was explored. A complete rupture of the superficial digital flexor tendon (SDFT) (at the musculo-tendinous junction) and *flexor carpi ulnaris* tendon (FCUT) was observed along with the loss of tendon tissues.

Case 2 was a four-year-old intact male crossbreed dog weighing 4 kg referred from the Local Animal Hospital for correction of tibial tuberosity transposition failure. The dog had a history of surgical correction of medial patellar luxation in the right stifle 10 days previously. Physical examination revealed a non-weight bearing lameness on the affected limb along with swelling of the stifle. Loose screw, broken tibial tuberosity and soft tissue swelling were detected on the survey radiograph (Figure 1).

Surgical correction was decided for in both cases. Both the dogs were premedicated with aceproma-



Figure 1. Preoperative radiograph showing a loose screw, broken tibial tuberosity and soft tissue swelling in Case 2

zine (Inj Sedazect[®], Sam Woo, Korea) 0.03 mg *i.m.* and butorphanol (Inj Butorphan[®], Myeon Moon Pharm, Korea) 0.2 mg *i.m.* Anaesthesia was induced using propofol (Inj Proviave[®], Myeon Moon Pharm, Korea) 6 mg *i.v.* and maintained with isofulroane and oxygen delivered through a cuffed endotracheal tube.

In Case 1, an incision along the laceration site was given and extended. After exploration of the wound retraction of the muscle-tendon unit was observed. The ruptured ends of both the tendons (SDFT and FCUT) were isolated, debrided and the uneven edges were excised (5 mm) to establish healthy tendon margins. The retraction of the muscle-tendon unit as well as excision of the irregular edges rendered it difficult to achieve apposition of the ruptured tendon ends. The tendons were released from the surrounding tissue to gain length and decrease tension. The ruptured SDFT was apposed with a 2-0 polyester suture using a modified Kessler suture pattern which was then followed by reinforcement with interrupted horizontal mattress sutures with 3-0 monofilament polydioxanone. After that a suturing gap formation was noticed on the carpus extension. To minimise this and provide additional strength, a fresh frozen Achilles tendon allograft (FFATA) was implanted to augment the injured tendons. The FFATA was thawed in crystalloid fluid at room temperature and cut (1 × 4 cm) so as to cover one third of the circumference of the tendon. Then, the FFATA was sutured using 3-0 monofilament polydioxanone in a simple interrupted pat-



Figure 2. Intraoperative photograph showing the superficial digital flexor tendon (thin black arrow) reconstruction using suture followed by fresh frozen Achilles tendon augmentation (black arrow) and ruptured *flexor carpi ulnaris* tendon (white arrow) in Case 1

tern 2 cm below the distal end of the SDFT rupture. The allograft was pulled toward the proximal end of the SDFT rupture with tension and anchored to the host tendon with the placement of simple interrupted sutures (Figure 2). The ruptured *flexor carpi ulnaris* tendon (FCUT) was reconstructed in the same manner. The wound was irrigated, and subcutaneous tissue and skin were closed over a penrose drain. The wound dressing was changed daily, and the drain was removed after two days.

A cranial and caudal splint, which had been contoured to the normal contralateral side, was placed from the paw to the elbow with a modified Robert-Jones bandage for four weeks. After that the splint was replaced with a Robert Jones bandage which remained in place for another two weeks. After splint removal, passive range of motion and stretching were initiated for 30 min daily for two weeks. No further external support was provided and progressive leash walk was started from six weeks postoperatively.

In Case 2 a lateral parapatellar longitudinal incision along the previous incision was made to explore the stifle. Partial rupture of the patellar tendon along with extensive tissue damage was noticed (Figure 3). The tibial tuberosity was found to be broken and the distal periosteal attachment was almost detached. There was also a fibrotic area around the loose screw. Because of the smaller size of the fragment it was not possible to fix the tibial tuberosity to the tibia. Therefore, a transverse bone tunnel was created through the tibia caudal to the cut tibial tuberosity using a small pin. A Krackow suture was placed in the patellar tendon, passed through the bone tunnel, and secured in place. The



Figure 3. Photograph showing damaged patellar tendon in Case 2



Figure 4. Augmentation of the patellar ligament after primary repair with fresh frozen Achilles tendon allograft in Case 2

FFATA cut to sufficient size $(1 \times 4 \text{ cm})$ was used to cover the patellar tendon. A polyester suture (2-0) was placed through the distal part of patellar tendon, passed through the tibia bone tunnel to attach the allograft tissue to the bone, and secured in place. The allograft was pulled toward the proximal patellar tendon and sutured in tension with the stifle joint in full extension. The belly of the FFATA was anchored to the patellar tendon using buried sutures. The edge of the allograft was sutured to the lateral and medial retinaculum and quadriceps tendon with 3-0 monofilament polydioxanone (Figure 4). The subcutaneous tissue and skin were closed in a routine manner.

Postoperatively, the stifle joint was immobilised in a modified Robert Jones bandage with a splint rod in full extension for four weeks. After that, the bandage was replaced with a soft padded bandage which was maintained in place for two more weeks. At six weeks postoperatively, no further external support was provided, and gradually increasing exercise was allowed.

Postoperatively, enrofloxacin (Inj Baytril 50[®], Bayer, Korea, 5 mg/kg *i.m.* every 12 h for two weeks) and tramadol (Zipan[®], Dong Jin Pharm, Korea, 2 mg/kg *p.o.* every 12 h for one week) were administered. In both cases, the surgical site healed without complications. There was no infection or clinical signs consistent with an immune response such as an exaggerated inflammation, exudation, swelling, or skin erythema (Figure 5). A good incorporation of the FFATA was noted in the radiograph obtained two months postoperatively in Case 2 (Figure 6). Both the dogs had normal ambulation and weight



Figure 5. Photograph six weeks postoperatively showing good healing of the surgical site with no clinical signs of immune response in Case 1

bearing on the affected limb 12 weeks postoperatively. In Case 2, there was a normal range of stifle motion (120°) and the thigh girth measurement was also similar to the contralateral limb. No postoperative complications were observed during a one-year follow up period except for slight contracture of the carpus and digits of the affected limb in Case 1.



Figure 6. A good incorporation of the fresh frozen Achilles tendon allograft noted in the radiograph obtained two months postoperatively in Case 2

DISCUSSION AND CONCLUSIONS

The tendon is stronger per unit area than muscle and its tensile strength is similar to that of bone, though it is flexible and slightly extensible (James et al. 2008). Reestablishment of tendon fibre continuity and the gliding mechanism is of great importance for tendon repair or replacement. Any injury to the tendon initiates several signalling events that recruit fibroblasts and stimulate the local tenocytes to synthesise collagen and extracellular components, establishing physical continuity and restoring partial or close-to-normal function of the tendon. The tendon repair process is usually very slow and characterised by scar formation and adhesions which in turn impede tendon gliding and have a negative impact on flexor tendon repair (James et al. 2008). Owing to the specialised nature of tissues, tendon and ligament regeneration has proved as an elusive goal for tissue engineering. There is a significant clinical need for augmentative and substitutional approaches that enhance the structural performance of damaged and degenerated tendon and ligament tissues (Kewa et al. 2011). To improve the results of tendon repair, the development of treatment modalities that may stimulate tissue healing, tendon gliding, mechanical strength, and return to normal function while preventing tendon gapping, ruptures, and extensive adhesions should be the aim.

Patellar tendon ruptures are fortunately rare injuries in both veterinary and human medicine (Falconiero and Pallis 1996; McNally et al. 1998; Shipov et al. 2008). The patellar tendon is the final connection of the extensor mechanism from the inferior pole of the patella to the tibial tubercle. Technically, it is a ligament (connecting bone to bone), but it has historically been referred to as a tendon because the patella is a sesamoid bone (Falconiero and Pallis 1996). The patella and the patellar tendon transmit the contraction of the quadriceps muscle to the tibia, which causes extension of the stifle joint. Rupture of the patellar tendon may be due to direct trauma or a sudden flexion of the knee coinciding with contraction of the quadriceps. Systemic disorders, chronic local stress and local or systemic administration of steroids have been proposed as possible causes of structural abnormalities which may predispose the patellar tendon to rupture (Shipov et al. 2008). Tendon rupture can also result from excessive stress during healing (James et al. 2008). However in Case 2, the partial rupture of the patellar tendon is thought to be due to the surgical trauma induced during the correction of medial patellar displacement in the local animal hospital.

Tendon grafts, whether autogenous or allogenic, undergo a more or less similar process of integration characterised by graft necrosis, revascularisation, cell repopulation and remodelling. The process of allograft integration is significantly affected by the method of tissue processing. The fresh allograft tissue is not suitable for implantation because it is highly immunogenic. The processes of freshfreezing, freeze-drying or cryopreserving allograft tissues significantly reduce the immunogenicity of the tissue by killing fibroblasts. These processes remove the loci for the major histocompatibility antigens, allowing allografts to be used even in immunologically incompatible hosts without provoking a significant immune response (Robertson et al. 2006). We used fresh frozen Achilles tendon (FFATA) for augmentation of the primary suture repair of SDFT and FCUT in one dog and patellar tendon in the other dog. Postoperatively, the surgical site healed without complications and there were no infection or clinical signs consistent with an immune response (exaggerated inflammation, exudation, swelling, or skin erythema).

In complex ligament injuries, tendon allograft provides a distinct advantage over autograft because of the limited availability of intact host tissue. The Achilles tendon (tendo calcaneus communis) is the strongest tendon in the structure of the musculoskeletal system in the dog. In human medicine, Achilles tendon allograft (ATA) has been used to successfully augment the injured patellar tendon (McNally and Marcelli 1998; Burnett et al. 2006). The ATA plays an important role in reconstruction of the extensively damaged tendon and ligament in a number of anatomical sites as it provides a long and wide aponeurosis (Robertson et al. 2006). In these cases, it was not possible to collect autogenous tissues because of extensive tissue injury and limited ability of the host tissue. That was why it was decided to perform the augmentation using FFATA which was found to be useful for the collection of sufficient amount of tissues as needed.

The successful repair of a ruptured tendon is challenging because of the tension during muscle contraction (James et al. 2008). Furthermore, due to the retraction of the muscle-tendon unit or extensive damage to the tendon tissue, there is a poor suture holding power at the repair site because of the lack of crossing collagen which also leads to a

decrease in tensile strength. These factors contribute to the gap formation or suture failure that have been associated with adhesion formation, poor clinical outcomes and tendon re-rupture. To minimise these risks, tendon repair may benefit from augmentation, especially if there is increased tension due to muscle contraction or loss of tendon length due to initial trauma or debridement. Augmentation of a ruptured tendon can help reduce gap formation by providing additional stability which counters excessive tension on the primary repair. In Case 2, the patellar tendon was found to be partially ruptured and friable which was accompanied by extensive tissue damage from the previous surgery. In this case, augmentation with FFATA after primary suture repair resulted in a good prognosis during a one year follow up period which is in agreement with a previous report in humans (McNally and Marcelli 1998).

The findings of this study suggest that ruptured tendons can be successfully repaired using suture followed by augmentation with FFCTA. Augmentation with FFATA may provide additional stability, which counters tension on the primary repair and reduces the chance of gap formation or suture failure in cases of reconstruction of damaged tendons and ligaments in dogs.

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